Examining the Role of Environmental Stress in the Etiology of Skeletal Defects

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Introduction
A variety of skeletal anomalies, especially cranial and dental traits, have been utilized as indicators of genetic affinity in biological distance studies and intracemtery kinship analyses. This type of research is founded on the assumption that anomalous traits have a high degree of heritability. Clinical studies, however, have clearly demonstrated that certain environmental factors, particularly dietary deficiency and disease, may trigger or enhance the genetic predisposition for developmental defects. As such, it is necessary to establish a clearer understanding of the precise etiology of these traits by examining various environmental influences.

Research Objectives
- Do nutrient deficiencies and/or disease stress cause elevated levels of skeletal defects?
- If so, which particular traits are most sensitive to environmental stress?

Background - Clinical Research
Teratogens & observed defects:
- Folic Acid (B₉), Riboflavin (B₂), Vitamin A, Vitamin E deficiencies
  → spina bifida, anencephaly, cleft palate, occipital vertebrae, vertebral blocks and clefting, brachydyactyly, polydactyly, coalitions, ossification abnormalities of the metacarpals, metatarsals, and phalanges,
- Hyperthermia (febrile illnesses)
  → brachydyactyly, polydactyly, cleft palate, vertebral anomalies

Materials & Methods
- 3 Arikara sites
  → Mobridge (N=135): 1600 – 1650 AD
  → Larson (N=193): 1679 – 1733 AD
  → Leavenworth (N=87): 1800 – 1832 AD
- Previous research suggests that:
  → Mobridge and Leavenworth experienced higher levels of stress than Larson
- 46 skeletal anomalies scored (present or absent)
  → Defects of the cranium, dentition, vertebral column, upper limb, hands, and feet
- Fisher’s Exact (1-tailed) to compare frequencies

Results
- 3 defects (os styloideum, asterion bone, occipital vertes) are more frequent in the stressed samples
- 1 defect (sacral shifting) more prevalent at Mobridge than at Leavenworth (Table 1, Figure 1)

Discussion and Conclusions
- These defects may be more sensitive to dietary and/or disease stress than other traits
  → vertebral column highly sensitive area for defects according to clinical literature
- However, at α=0.05 (46 traits) approximately 2 significant differences are expected by random chance alone
  → suggests a stronger genetic component for most traits, with little environmental influence
- The results of this study support the use of skeletal anomalies as indicators of genetic affinity

Table 1. Selected defect frequencies and statistical comparisons

<table>
<thead>
<tr>
<th>Skeletal Anomaly</th>
<th>Site Frequency (%)</th>
<th>Comparisons (p-val)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MO</td>
<td>LA</td>
</tr>
<tr>
<td>Os Styloideum</td>
<td>18.3</td>
<td>4.6</td>
</tr>
<tr>
<td>Asterion Bone</td>
<td>4.9</td>
<td>8.6</td>
</tr>
<tr>
<td>Occipital Vertebrae</td>
<td>12.7</td>
<td>4.1</td>
</tr>
<tr>
<td>Sacral Shifting</td>
<td>35.1</td>
<td>21.8</td>
</tr>
</tbody>
</table>

* Bolded red text indicates statistically significant difference (α=0.05)

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Selected References